

reader, but without benefit and without harm. In some cases small doses have been badly borne, but on increasing the dose the unpleasant symptoms have disappeared.—*Boston Med. and Surg. Jour.*, No. 12.

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LOCOMOTOR ATAXY, WITHOUT DISEASE OF THE POSTERIOR COLUMNS OF THE SPINAL CORD.—Dr. A. Hughes Bennett read notes of this case before the Clinical Soc. of London, Feb. 27th. The patient during life presented all the prominent symptoms of tabes dorsalis, while after death the posterior columns and cornua of the spinal cord were found without a trace of disease. From this fact, important physiological and pathological conclusions were drawn. The patient, a man, aged forty-eight, suffered from all the usual symptoms of locomotor ataxia, including inco-ordination of movement, without loss of muscular power, a typical ataxic gait, Romberg's symptom, impaired and retarded sensibility of the lower extremities, lancinating pains, and loss of the knee-jerk phenomena. After death evidences of general acute cerebritis were found, with patches of recent softening. In the medulla oblongata, there was a mass of sarcomatous infiltration occupying its posterior and central aspects; with the exception of one anterior cornu in a limited portion of the lumbar region, the spinal cord was perfectly normal throughout, as were also the posterior cornu, and roots within the circle of the spinal membranes. Outside these the posterior roots in the dorsal and lumbar regions were found involved in a mass of sarcoma, which extended as far upwards as the cervical portion of the cord. In the lumbar region the anterior roots were implicated, but only to a very limited degree. Microscopic preparations and drawings illustrating these facts were shown at the meeting. In commenting on this fact, special attention was directed to the pathological and clinical phenomena. It was pointed out that the patches of central softening were probably quite recent, and due to the acute cerebritis, which supervened shortly before death. The sarcomatous growth in the medulla was stated to be a rare pathological condition; and the absence of pronounced bulbar symptoms was explained by the supposition that the morbid material had infiltrated the normal structures, without causing their serious degeneration. Although one anterior horn was implicated, and some of the anterior roots slightly involved in the disease, it was evident that this had caused few symptoms during life, as the motor power was unimpaired, there was no muscular wasting, and the retinal reactions were normal. The clinical point illustrated by this case, to which it was the object of the paper specially to direct attention, was the relation which existed between the spinal symptoms and the lesions in the posterior roots of the cord. It was pointed out that the patient during life suffered from all the prominent symptoms of tabes dorsalis, and presented a fairly typical clinical picture of what was understood of that disease. It was then stated that the universal belief was that the essential lesion of locomotor ataxy

was sclerosis of the posterior root-zones of the cord. In the case under consideration there was no trace of disease of any portion of the posterior columns, proving that, for the production of ataxy, degeneration of the cord was not an essential factor. Reference was made to the cases published by M. Déjerine, in which there were all the symptoms of tabes, and after death only parenchymatous neuritis of the peripheral nerve-endings found, the cord-roots being intact. From this it was maintained that interruption of the different paths, at the periphery, was capable of causing ataxy in as characteristic a manner as when these tracts were diseased in the cord. The case under notice showed that lesion of a third locality, namely, of the posterior spinal roots, might produce the same effects. It was asserted that this was a pathological confirmation of the experimental researches of Van Deen and Claude Bernard, who, by dividing the posterior roots, induced inco-ordination of movements without motor paralysis. It supported the view that those elements which conveyed the impulses regulating co-ordination, were situated in the afferent paths of the nervous system. Whatever theory be advanced to explain the physiology of locomotor ataxy, this case served to show that the point at which, in the causation of the phenomena, the nervous path was interrupted, must not of necessity, as was generally asserted, be primarily situated in the posterior root-zones of the spinal cord. Reflection on this fact suggested that the anatomical substratum of that protean disorder which was at present recognized under the term *tabes dorsalis*, had not yet been fathomed. The assemblage of symptoms probably consisted of a combination of different pathological conditions, many of which were represented by phenomena common to all, and each of which in time might be distinguished. It was possible that a case such as the present might prove as a link in the chain of discovery.—*Brit. Med. Jour.*, No. 1,262.

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CLONIC SPASMS OF UPPER LIMBS.—Dr. C. E. Beevor read a paper before the Medical Society, London, on clonic spasm of the upper limbs, with anæsthesia of the left side, in a girl aged 16, who had scarlet-fever ten years before she came under observation, and, six months after the fever, began to have twitching of the head and face to the left, and movements in both arms. She came under treatment by Dr. Barlow for choreiform movements affecting all the limbs seven years ago; movements had persisted ever since. A year ago, the left arm alone was affected with pronation, extension of the elbow, and adduction of the shoulder, but voluntary movements were possible; she also had occasional rapid twisting of the head, and both eyes were turned to the left. In October, 1884, the right arm was affected in a similar way, and the left arm became anæsthetic and analgesic. When the patient was shown at the meeting, the movements of the left arm were much quieter, but the limb was almost completely paralyzed, and